WHAT IS CLAIMED IS:

1. A method for determining whether a subject has or is predisposed to developing an arterial restenosis, comprising detecting a restenosis associated allele in a nucleic acid sample from the subject, wherein detection of the restenosis allele indicates that the subject has or is predisposed to the development of a restenosis.

2. A method of claim 1, wherein the restenosis allele is selected from the group consisting of allele 1 of any of the following markers: IL-1A (+4845), IL-1B (-511), IL-1B (+3954) and IL-1RN (+2018) or an allele in linkage disequilibrium therewith.

3. A method of claim 1, wherein said detecting step is selected from the group consisting of:

a) allele specific oligonucleotide hybridization;
b) size analysis;
c) sequencing;
d) hybridization;
e) 5' nuclease digestion;
f) single-stranded conformation polymorphism;
g) allele specific hybridization;
h) primer specific extension; and
j) oligonucleotide ligation assay.

4. A method of claim 1, wherein prior to or in conjunction with detection, the nucleic acid sample is subject to an amplification step.

5. A method of claim 2, wherein said amplification step employs a primer pair selected from the group consisting of any of SEQ ID Nos. 1 and 2; 3 and 4; 5 and 6; 7 and 8; 9 and 10; 11 and 12; and 13 and 13 and 14.

6. A method of claim 3, wherein said size analysis is preceded by a restriction enzyme digestion.

7. A method of claim 6, wherein said restriction enzyme digestion uses a restriction enzyme selected from the group consisting of Alu I, Msp I, Nco I, Fnu 4HI, Ava I, Bsu 36 I, and Taq I.

8. A kit for determining the existence of or a susceptibility to developing a restenosis in a subject, said kit comprising a first primer oligonucleotide that hybridizes 5′ or 3' to an allele selected from the group consisting of allele 1 of any of the following markers: IL-1A (+4845), IL-1B (-511), IL-1B (+3954), IL-1RN (VNTR) and IL-1RN (+2018) or an allele in linkage disequilibrium therewith.

9. A kit of claim 8, which additionally comprises a second primer oligonucleotide that hybridizes either 3' or 5' respectively to the allele so that the allele can be amplified.

10. A kit of claim 9, wherein said first primer and said second primer hybridize to a region in the range of between about 50 and about 1000 base pairs.

11. A kit of claim 8, wherein said primer is selected from the group consisting of any of SEQ ID Nos. 1-14.

12. A kit of claim 8, which additionally comprises a detection means.

13. A kit of claim 12, wherein the detection means is selected from the group consisting of:

a) allele specific oligonucleotide hybridization;
b) size analysis;
c) sequencing;
d) hybridization;
e) 5' nuclease digestion;
f) single-stranded conformation polymorphism;
g) allele specific hybridization;
h) primer specific extension; and
j) oligonucleotide ligation assay.

14. A kit of claim 8, which additionally comprises an amplification means.

15. A kit of claim 8, which further comprises a control.

16. A method for selecting an appropriate therapeutic for an individual that has or is predisposed to developing a restenosis, comprising the steps of: detecting whether the subject contains a restenosis associated allele and selecting a therapeutic that compensates for a restenosis causative functional mutation that is in linkage disequilibrium with the restenosis associated allele.

17. A method of claim 16, wherein said detecting is performed using a technique selected from the group consisting of:

a) allele specific oligonucleotide hybridization;
b) size analysis;
c) sequencing;
d) hybridization;
e) 5' nuclease digestion;
f) single-stranded conformation polymorphism;
g) allele specific hybridization;
h) primer specific extension; and
j) oligonucleotide ligation assay.

18. A method of claim 16, wherein prior to or in conjunction with detecting, the nucleic acid sample is subjected to an amplification step.

19. A method of claim 18, wherein said amplification step employs a primer selected from the group consisting of SEQ ID Nos. 1-14.

20. A method of claim 17, wherein said size analysis is preceded by a restriction enzyme digestion.

21. A method of claim 20, wherein said restriction enzyme digestion uses a restriction enzyme selected from the group consisting of Alu I, Msp I, Nco I, Fnu 4HI, Ava I, Bsu 36 I, and Taq I.

22. A method of claim 21, wherein the restenosis therapeutic is selected from the group consisting of: an agent that suppresses the development of a hyperplasia and an agent that directly inhibits cellular growth.

23. A method of claim 22, wherein the agent that suppresses the development of a hyperplasia is selected from the group consisting of a lipid lowering drug, an antiplatelet agent, an anti-inflammatory agent, an antihypertensive agent and an anticoagulant.

24. A method of claim 21, wherein the restenosis therapeutic is a modulator of an IL-1 activity.

25. A method of claim 24, wherein the IL-1 activity is IL-1α.

26. A method of claim 24, wherein the IL-1 activity is IL-1β.

27. A method of claim 24, wherein the IL-1 activity is IL-1RN.

28. A method of claim 24, wherein the modulator of an IL-1 activity is a protein, peptide, peptidomimetic, small molecule, nucleic acid or a nutraceutical.

29. A method of claim 24, wherein the modulator is an agonist.

30. A method of claim 24, wherein the modulator is an antagonist.

31. A method of claim 16, wherein the restenosis associated allele is selected from the group consisting of: allele 1 of any of the following markers: IL-1A (+4845), IL-1B (-511), IL-1B (+3954) and IL-1RN (+2018) or an allele in linkage disequilibrium therewith.

32. A method of claim 16, wherein the restenosis causative functional mutation is an allele of IL-1B (+6912), IL-1B (-511) or IL-1RN (+2018).

33. A method for determining the effectiveness of treating a subject that has or is predisposed to developing restenosis with a particular dose of a restenosis therapeutic, comprising the steps of:

a) detecting the level, amount or activity of an IL-1 protein; or an IL-1 mRNA or DNA in a sample obtained from a subject;

(b) administering the particular dose of the particular therapeutic to the subject; detecting the level, amount or activity of an IL-1 protein; or an IL-1 mRNA or DNA in a sample obtained from a subject; and

(c) comparing the relative level, amount or activity obtained in step (a) with the level, amount or activity obtained in step (b).

34. A method of claim 33, wherein the therapeutic is selected from the group consisting of: an agent that suppresses the development of a hyperplasia or an agent that directly inhibits cellular growth.

35. A method of claim 34, wherein the agent that suppresses the development of a hyperplasia is selected from the group consisting of; a lipid lowering drug, antiplatelet agent, an anti-inflammatory agent; an antihypertensive agent and an anticoagulant.

36. A method of claim 33, wherein the therapeutic is a modulator of an IL-1 activity.

37. A method of claim 36, wherein the IL-1 activity is IL-1α.

38. A method of claim 36, wherein the IL-1 activity is IL-1β.

39. A method of claim 36, wherein the IL-1 activity is IL-1RN

40. A method of claim 34, wherein the therapeutic is a protein, peptide, peptidomimetic, small molecule or a nucleic acid.

41. A method of claim 36, wherein the modulator is an agonist.

42. A method of claim 36, wherein the modulator is an antagonist.

43. A method for treating or preventing the development of a restenosis in a subject comprising the steps of detecting the presence of a restenosis associated allele and administering to the subject a therapeutic that compensates for causative mutation that is in linkage disequilibrium with the restenosis associated allele.

44. A method of claim 43, wherein the detecting step is selected from the group consisting of:

a) allele specific oligonucleotide hybridization;
b) size analysis;
c) sequencing;
d) hybridization;
e) 5' nuclease digestion;
f) single-stranded conformation polymorphism;
g) allele specific hybridization;
h) primer specific extension; and
j) oligonucleotide ligation assay.

45. A method of claim 43, wherein prior to or in conjunction with detecting, the nucleic acid sample is subjected to an amplification step.

46. A method of claim 45, wherein said amplification step employs a primer selected from the group consisting of any of SEQ ID Nos. 1-14.

47. A method of claim 44, wherein said size analysis is preceded by a restriction enzyme digestion.

48. A method of claim 47, wherein said restriction enzyme digestion uses a restriction enzyme selected from the group consisting of Alu I, Msp I, Nco I, Fnu 4HI, Ava I, Bsu 36 I, and Taq I.

49. A method of claim 43, wherein the therapeutic is selected from the group consisting of: an agent that suppresses the development of a hyperplasia or an agent that directly inhibits cellular growth.

50. A method of claim 49, wherein the agent that suppresses the development of a hyperplasia is selected from the group consisting of; a lipid lowering drug, antiplatelet agent, an anti-inflammatory agent, an antihypertensive agent and an anticoagulant.

51. A method of claim 43, wherein the therapeutic is selected from the group consisting of: a modulator of an IL-1 activity.

52. A method of claim 51, wherein the IL-1 activity is IL-1α.

53. A method of claim 51, wherein the IL-1 activity is IL-1β.

54. A method of claim 51, wherein the IL-1 activity is IL-1Ra.

55. A method of claim 51, wherein the therapeutic is a protein, peptide, peptidomimetic, small molecule or a nucleic acid.

56. A method of claim 51, wherein the modulator is an agonist.

57. A method of claim 51, wherein the modulator is an antagonist.

58. A method of claim 43, wherein the restenosis associated allele is allele 1 of any of the following markers: IL-1A (+4845), IL-1B (-511), IL-1B (+3954) and IL-1RN (+2018) or an allele in linkage disequilibrium therewith.

59. A method of claim 43, wherein the ILD causative functional mutation is IL-1B (+6912) allele 2, IL-1B (-511) allele 2 or IL-1RN (+2018) allele 2.

60. A method for screening for a restenosis therapeutic comprising the steps of:

a) combining an IL-1 polypeptide or bioactive fragment thereof, an IL-1 binding partner and a test compound under conditions wherein, but for the test compound, the IL-1 protein and IL-1 binding partner are able to interact; and

b) detecting the extent to which, in the presence of the test compound, an IL-1 protein/IL-1 binding partner complex is formed, wherein an increase in the amount of complex formed by an agonist in the presence of the compound relative to in the absence of the compound or a decrease in the amount of complex formed by an antagonist in the presence of the compound relative to in the absence of the compound indicates that the compound is a restenosis therapeutic.

61. A method of claim 60, wherein the agonist or antagonist is selected from the group consisting of: a protein, peptide, peptidomimetic, small molecule or nucleic acid.

62. A method of claim 61, wherein the nucleic acid is selected from the group consisting of: an antisense, ribozyme and triplex nucleic acid.

63. A method of claim 60, which additionally comprises the step of preparing a pharmaceutical composition from the compound.

64. A method of claim 60, wherein the IL-1 polypeptide is IL-1α.

65. A method of claim 60, wherein the IL-1 polypeptide is IL-1β.

66. A method of claim 60, wherein the IL-1 polypeptide is IL-1Ra.

67. A method for identifying a restenosis therapeutic, comprising the steps of:

a) contacting an appropriate amount of a candidate compound with a cell or cellular extract, which expresses an IL-1 gene; and

b) determining the resulting protein bioactivity, wherein a decrease of an agonist bioactivity or a decrease in an antagonist bioactivity in the presence of the

compound as compared to the bioactivity in the absence of the compound indicates that the candidate is a restenosis therapeutic.

68. A method of claim 67, wherein the modulator is an antagonist of an IL-1α or an IL-1β, bioactivity.

69. A method of claim 67, wherein the modulator is an agonist of an IL-1RN bioactivity.

70. A method of claim 67, wherein in step (b), the protein bioactivity is determined by determining the expression level of an IL-1 gene.

71. A method of claim 70, wherein the expression level is determined by detecting the amount of mRNA transcribed from an IL-1 gene.

72. A method of claim 70, wherein the expression level is determined by detecting the amount of the IL-1 product produced.

73. A method of claim 70, wherein the expression level is determined using an anti-the IL-1 antibody in an immunodetection assay.

74. A method of claim 70, which additionally comprises the step of preparing a pharmaceutical composition from the compound.

75. A method of claim 70, wherein said cell is contained in an animal.

76. A method of claim 75, wherein the animal is transgenic.

77. The method of any of claims 1, 16 or 43, wherein the presence of an IL-1 locus allelic pattern comprising allele 1 of each of IL-1A (+4845), IL-1B (+3954), IL-1B (-511), and IL-1RN (+2018), is detected.

78. The method of claim 77, further comprising determining whether allele 1 of IL-1RN(+2018) is carried in the homozygous state.

79. A method of claim 1, wherein the restenosis allele is selected from the group consisting of allele 1 of any of the following markers: IL-1A (+4845), IL-1B (-511), IL-1B (+3954) and IL-1RN (+2018) or an allele in linkage disequilibrium therewith.